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SURGICAL LESIONS

OF

THE BRAIN AND ITS ENVELOPES.

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GENTLEMEN: I shall call your attention to-day to an extremely important subject, the surgical diseases of the brain and its envelopes—lesions attributable directly to traumatism and the consecutive lesions following injury to the skull, the brain, and its envelopes. You will recognize the importance of secondary lesions of the envelopes if I call your attention to the fact that any lesion of the meninges is liable to extend to the brain substance proper by continuity of the inflammatory process, or may prove fatal by compression of the brain by the products of inflammation, which takes place on account of the unyielding character of the cranial vault. I wish you to remember that wounds of the brain, both incised and contused, may heal and cicatrize in a manner similar to that noted after injuries of other organs; in this respect we differ with the older authorities, who believed that injuries of the brain substance proper were incapable of repair; a loss of continuity in the brain substance proper which does not suspend an essential function may be followed by cicatrization and complete repair, and the organ may be restored to its functional integrity, provided, however,

that the injury is unattended by consecutive traumatic infection, and consequently unattended by secondary inflammatory lesions; in other words, the loss of substance is replaced by a reconstructive process from the normal histological elements of the brain initiated by the traumatism.

About thirty years ago an eminent German physiologist (Wagner) made a series of experiments to ascertain the capacity of the brain to repair itself, and with another view to ascertain the physiological function of different portions of the brain. He found on removing sections of different portions of the organ that the operation, as a rule, was not followed by a process of destructive inflammation—that cicatrization was established in the same manner as when the injury is inflicted upon some other organ of less physiological importance. He not only found that wounds of the brain healed, but at the point of injury loss of brain substance was liable to occur as a result of consecutive cicatricial contraction without causing serious functional impairment. He ascertained this fact by a series of experiments, weighing different portions of the brain before and after the injury so accurately as to find the fraction of a milligramme of difference. Again, you will remember the classical case reported by Bigelow, of Boston, in 1855, where a crowbar passed through the brain substance, entering the skull from below upward, producing not only a great loss of brain substance, but contusion of the adjacent portions—a condition which must of necessity result in extensive cicatricial contraction during the reparative process. If, as was here the case, no loss of function occurred after the wound had healed, you can readily imagine that even an extensive loss of brain tissue does not necessarily interfere with the normal physiological functions of the brain if the injury is not followed by a secondary destructive process.

In introducing the subject of surgical lesions of the brain as they follow injuries of the cranium or its contents, it is necessary for a moment to direct your attention to those rare affections which occasionally follow incomplete fractures of the skull. By such an injury I mean a contusion affecting the cancellated structure between the external and internal table of the cranial bones; an indentation of one or more of the cranial bones by external violence, which is subsequently restored by means of the elasticity of the bones, assisted by the intracranial pressure, but resulting in compression of the cancellated tissue between the tables. It is a form of incomplete fracture as we observe it in the skull, the result of compression, which produces a condensation of the cancellated tissue, but does not affect the external or the internal table, an injury not recognizable by external signs, because the external table is smooth, while the internal table has a sufficient amount of elasticity to restore the normal relation of the parts though the diploë be crushed. It is a partial crushing of the diploëic structure, the result of force applied from without, as I have previously informed you, and which may temporarily indent the skull to the extent of even half an inch without producing fracture of the compact layers of the bone. This variety of fracture can only be recognized by evidences resulting from extravasation of blood, and ecchymosis at the point of injury, and by the immediate effects of the injury upon the functions of the brain. Its existence is also suggested a certain length of time after the injury has been received by a consecutive formative periostitis—a periostitis induced directly by the traumatism, resulting in the production of new bone, provided no infection has taken place at the seat of injury.

It is important to remember this form of injury from the fact that occasionally we meet with cases of injury

of the cranial bones subsequently followed by serious brain disease, the secondary brain lesion being the direct result of the local injury in the diploë; an injury producing, in the first place, a laceration of the small veins in the diploë followed by thrombosis, the thrombus acting as a favorable culture soil for germs that may be accidentally introduced from a slight abrasion, resulting in an osteomyelitis—the osteomyelitis, again, extending by means of the consecutive thrombophlebitis to the subjacent meninges and to the brain itself. These remarks should teach you simply this fact: that all injuries of the skull, even when apparently slight, should put you on your guard with reference to the management of the case. Even a comparatively slight contusion of the cranial vault may be followed by a complexus of symptoms indicative of a grave central lesion. It is on this account that such injuries, slight as they may appear, should receive most careful treatment. This should have for its aim the prevention of secondary lesions; consequently, it must be directed toward the prevention of the inflammatory processes in the bone, and their extension to the brain and its envelopes. Rest in the elevated position, and cold applications are the measures upon which you rely, in the first place, to prevent an undue local inflammation, and if evidences of osteomyelitis appear, as evidenced by the appearance of fever, pain at the seat of injury, local œdema, then resort to early operative measures to reach the inflammatory focus, etc., before the infective thrombophlebitis has had time to extend to the subjacent brain tissue.

In speaking of traumatic meningitis, it is necessary to recognize two distinct varieties, the primary and the secondary. The classification introduced as early as the time of Ambrose Paré, and since recognized by all surgical authors, is this: A primary meningitis is that which is produced directly by traumatism following an

injury within twenty-four to seventy-two hours; a secondary meningitis, on the other hand, is an inflammation of the meninges occurring an indefinite period of time after the local injury, but where the primary injury bears a direct etiological relation to the secondary lesion. When I call your attention to the causes which produce these two varieties, the distinction will appear plain to you. By a primary meningitis, I mean that form which results directly from an absorption of the products of putrefaction at the point of injury; a meningitis following a compound fracture of the skull, for instance, exposing the dura mater directly to infection. If the dura mater is exposed directly to the products of putrefaction, the septic germs penetrate the substance of the meninx proper, and the inflammation extends in a central direction *pari passu* with the invasion of the primary cause. The process begins at the point of injury, extends in all directions from a common central point toward the circumference, and in a central direction, by continuity and contiguity. By extension by continuity, I mean that the infective principle pervades the tissues of the dura mater, extending deeper and deeper, involving the subjacent envelopes, the arachnoid, and pia mater, and producing an inflammatory process in these membranes resembling in every respect the character of the inflammation in the dura mater, constituting the lepto-meningitis of recent pathologists. Again, extension takes place by contiguity of tissue, the pia mater being in close contact with the cortical layer of the brain the process extends from the meninges to the brain, so that we have a meningitis involving all of the meninges accompanied with an encephalitis, or a meningo-encephalitis, meaning an inflammation of all the meninges of the brain with inflammation of the subjacent brain tissue. Consequently, we again assert that, as in any other locality, a destructive inflammation of the

meninges of the brain and the subjacent brain tissue following an injury is always to be considered as a direct result of traumatic infection or traumatism combined with infection from some antecedent pathological product. *No infection, no meningitis. No infection, no encephalitis.*

By a primary meningitis, then, we mean that which is induced by primary infection. This is important in a practical sense, inasmuch as it will again remind you of the importance of resorting to thorough local disinfection in all cases of compound fracture of the skull in order to obviate a consecutive meningitis. It should impress upon you again the fact that when a fracture is once compound you necessarily make it more compound in the attempt to secure free drainage. If you have an indented fracture which will not permit of free drainage between the fragments, it is necessary to expose the meninges by removing portions of the detached bone, and if the bone is not detached, you should enlarge the opening by means of a bone forceps or chisel, not only for the purpose of resorting to thorough primary disinfection underneath the fractured bone, but also for the purpose of insuring free drainage subsequently. We observe the same rule in these instances as we do in all other serous cavities. An infective agent, once introduced, is beyond our control if it has permeated the tissues beneath; consequently, the larger the opening the freer the drainage; the better the access to the infected district, for the application of disinfecting agents, the better the results.

By a secondary meningitis of a traumatic origin, I mean that which may follow an injury of the skull or the superimposed soft tissues at some indefinite subsequent time.

Premising, again, that, as in cases of primary meningitis, the secondary form is due to the same infective

cause, but that the infection has remained for a certain time latent, local, and has extended subsequently by the development of certain well-defined, local, pathological conditions—as, for instance, suppurative osteomyelitis, a condition to which I have previously alluded in cases of contusion of the cranial bones, as being the result of the introduction of specific germs which we have learned produce suppurative inflammation. This condition may not be apparent at first, but gradually, as the disease proceeds, it involves the adjacent veins, producing a thrombophlebitis in the diploë, the thrombus growing in a central direction until it reaches the meninges, where a similar suppurative process is initiated as at the primary seat of infection. It is a suppurative meningitis when the lesion is caused by the extension of suppurative inflammation from the exterior or as the result of a purulent thrombophlebitis. But the specific cause may have affected one of the large venous channels within the cranial vault producing a primary sinus-phlebitis, and the meningitis may be due to extension from this source. By this I mean an inflammation of the large venous sinuses in the cranial cavity, the result of extension of the inflammatory process from the local injury to a subjacent sinus. In considering this part of our subject you must remember the direct communication that exists between the external veins of the skull and diploë and the sinuses through the medium of connecting venous channels. If, for instance, we find we have to deal with only a local inflammation of an infective type, a furuncle, a suppurating wound of the soft parts, or an anthrax, and no fracture has existed, but a local infection has taken place in the soft tissues; if from this focus of infection a thrombophlebitis take place, the thrombus forms in the vein, growing by aggregation in a central direction, and gradually reaches the sinus, a thrombus is formed in the sinus,

and its presence provokes an inflammation in the sinus itself producing a sinus-phlebitis. By direct contact with the sinus of the meninges the inflammation extends in all directions, producing an infective meningitis resembling, in its character and results, in every respect the primary inflammation in the sinus and in the superficial veins. Furthermore, the infection may have a still deeper origin, and may extend in a peripheral instead of in a central direction; in other words, the inflammatory primary disease may originate within the brain, the process affecting the meningeal membranes from the centre of the brain in a peripheral direction, hence a consecutive meningitis, following a primary encephalitis terminating in an encephalo-meningitis.

In alluding to the symptomatology of traumatic meningitis, the first symptom you will observe is a changed condition of the mind of the patient; a psychical perturbation due to hyperæmia of the superficial cortical substance of the brain, as this portion of the brain is predisposed by the anatomical arrangement of its structure to extension of the inflammatory process from the meninges. The perverted sensorium points to an increased, exaggerated physiological function as the result of increased tissue changes in the gray matter of the cortex, due to an increased vascularization of that portion of the brain, producing increased exaltation of the psychical functions of the brain. If you are called upon to treat this disease as we occasionally meet with it—after a compound fracture of the skull, for instance—the first thing you will notice on your visit is that your patient is contrary, peevish, morose; complains of every little thing; the least noise produces painful sensations. As the disease progresses, or as long as this exaltation in the psychical function of the brain increases, delirium follows—delirium sometimes of an active character; the patient even attempts to run away, jump out of the

window, etc.; consequently, any serious change pointing toward a perverted sensorium in cases of compound fracture of the skull should receive your earnest and early attention. One of the first positive evidences of the existence of traumatic meningitis is an excruciating headache, referable at first to the primary seat of the inflammation, but as the process extends it becomes more diffuse. It is a headache which it is difficult to describe, but the words "excruciating" and "diffuse" are sufficient to indicate the character and extent of the local inflammation. It is a pain resulting from inflammation of a serous membrane dense in structure—a pain resulting from vascular engorgement and increased intracranial pressure. As a result of increased exaltation of the cerebral functions we very frequently observe convulsions; the area of peripheral irritation results in exaggeration of the motor function—convulsions general or local, as the case may be. If only one side of the brain is affected, the process is yet limited; in all probability the convulsive movements will occur on the opposite side. Again, if the process is limited to a particular sphere, involving the nerves supplying certain definite muscular groups, you observe a monospasm in the group of muscles supplied by the nerves which are irritated by the central disease.

Vomiting is an early symptom, and when persistent denotes an acute and severe attack. Persistent vomiting, in the absence of mechanical obstruction or inflammatory affections of the digestive tract, should always awaken suspicion of the existence of cerebral disease. Cerebral vomiting is not the retching due to a disturbance of the digestive function, but a mechanical vomiting, so that in the absence of gastric disturbance, and the rapidity with which the attacks occur, you may suspect a central cause. The pulse, in the beginning of the disease, is always increased in frequency, say from

80 to 120, full and bounding, until the local disturbance has led to an increase in intracranial pressure, when it becomes slow, indicating in this connection compression of the brain. By a slow pulse I mean one that varies from 50 to 60 beats per minute. After a few days it again becomes rapid and feeble, being simply the forerunner of approaching dissolution, indicating a general paralysis, due to the central cause; although at first rapid, it has a natural tendency to become softer and smaller in volume, consequently the slowness following a rapid pulse is not a favorable, but a serious change. When it becomes more rapid and small, it is an indication that general paresis is occurring as one of the results of cerebral compression.

The temperature in meningitis is always increased —usually 100° to 104° F.—the temperature usually can be considered as an index of the intensity and extent of the infection. As in all other infectious traumatic diseases, an early high rise in temperature forebodes danger. It simply means that the infection has taken place rapidly and has become diffuse; that the local process has extended with great rapidity, involving at an early date a large surface of the meningeal structures. A paresis in cases of meningitis is always the result of secondary pathological changes produced by the local inflammation; by this I mean either an increase in intracranial pressure, resulting in cerebral compression and consequently a localized area of anæmia in the brain, or an extension of the inflammatory process to the brain and destruction of the centre of sensation or motion, as the case may be; a paralysis first localized, extending as the local cause spreads, affecting more distant structures; a paralysis indicating simply the extent of the cerebral injury or disease, primary or secondary, according to the pathological condition which characterizes the case. The pupils in

meningitis are first, as a rule, contracted ; in a few days they may be normal, but on testing their contractility you will alway notice a slow response to light, which is one of the characteristic features of meningeal inflammation. As the disease progresses, and exudation takes place, and compression is produced, one pupil may be dilated and the other contracted. When cerebral compression is well marked, dilatation of both pupils takes place.

By pyæmia as a complication of traumatic meningitis we understand the existence of distant or metastatic abscesses, resulting from disintegration of a thrombus in the sinus, or in the veins.

We have now arrived at an important pathological consideration of cerebral pathology. Strange as it may appear, ancient authorities have entertained the most diverse opinions in regard to the relations existing between a local suppuration and distant abscesses. For instance, the distinguished pathologist, Bichat, claimed the existence of a peculiar sympathy between the brain and the liver; a sympathy which would come into activity in case of pathological conditions in the brain, producing similar conditions in the liver. Another writer claims that metastatic abscess of the liver is frequently a concomitant condition in cases of pyæmia from cerebral suppuration, and that the disease is first located in the liver, and subsequently extends from it to the brain. Recent pathological research has demonstrated that pyæmia in thrombophlebitis or sinus-phlebitis constitutes one of its greatest dangers, and on post-mortem examinations is found as one of the most frequent causes of death ; the thrombus by disintegration will produce pyæmia just as when a thrombus forms in any other part of the body under similar circumstances, giving rise to embolism and metastatic abscesses. In cases of suppurative inflammation of the

brain and its envelopes, the process is facilitated by the direct communication of the large reservoirs of the brain with the jugular veins, so that pyæmia as we observe it in cases of thrombophlebitis or sinus-phlebitis means simply the coexistence of metastatic abscesses caused by embolism from disintegration of the infected thrombus.

In the differential diagnosis we must take into consideration concussion. Concussion may precede meningitis, and may complicate the clinical picture. Concussion following immediately after an injury, produces symptoms not referable to any particular portion of the brain, but a condition in the entire cerebrum resulting in partial suspension for the time being of its physiological functions; consequently, during the first twenty-four or forty-eight hours it is impossible to make a positive differential diagnosis. If, however, the symptoms prove more permanent; if concussion is followed by meningitis, there is no restoration of function *ad integrum*, as in uncomplicated cases of concussion, but the process proceeds and results in the manifestation of focal symptoms. Traumatic meningitis is distinguished from incipient encephalitis by the absence of symptoms indicating central irritation—symptoms expressive of exaltation of the cerebral functions, if the inflammatory process in the brain is not complicated by meningitis from the beginning. Again, encephalitis, at first primary and localized, gives rise to early focal symptoms; that is to say, symptoms pointing toward a distinct portion of the brain as the seat of the lesion. The local injury, if unattended by meningitis, again gives rise to focal symptoms from the very beginning, localizing the lesion in the brain, and an absence of those severe symptoms pointing to cerebral irritation, as we observe it in meningitis.

Abscess in the brain has quite a different clinical history: contusion, thrombophlebitis, sinus-phlebitis,

cranial osteomyelitis preceding the formation of pus in the brain; it is also attended by focal symptoms, as evidenced by circumscribed regions of paralysis of certain definite muscular groups, pointing to the local lesion in the brain as the cause of the paralysis. Again, abscess of the brain is not attended by such a constant rise in temperature as is found in cases of traumatic meningitis—in fact, in many of the cases recorded the temperature has been noted as being subnormal.

It is impossible, in the beginning, to differentiate simple congestion from inflammation, inasmuch as inflammation is always preceded by congestion; but congestion independent of inflammation is only a temporary condition yielding to local or general measures.

In regard to the localization of the primary lesion a great deal has been said and written.

Bergmann made an attempt to differentiate between convex meningitis and basilar meningitis, and the result of his observations appears to prove that a convex meningitis is noted particularly for its acuity; for its rapid extension and the intensity of symptoms. It has also been claimed as a distinguishing feature that in cases of convex meningitis paralysis appears sooner and is more extensive. Basilar meningitis, on the other hand, is noted for the slowness of the local process, the absence of intense symptoms due to cortical irritation; circumscribed paralysis appearing at a late stage; again, it differs from meningitis of the convex surface of the brain by a tendency of the inflammation to extend along the spinal structures, being followed by a spinal meningitis, as indicated by rigidity of the muscles of the spine and neck, the disease resembling in this respect, in many instances, cerebro-spinal meningitis.

The prognosis has not yet been considered. It is sufficient to say that it is always grave; the majority of cases of traumatic infective meningitis prove fatal. The

prognosis is always unfavorable because the infection once thoroughly established is beyond the control of the surgeon ; the process always manifests an inherent tendency to extend to all of the membranes and the subjacent brain tissue, and even to the contents of the spinal canal. The prognosis is grave because traumatic meningitis means a suppurative meningitis; suppuration necessarily follows from the very nature of the primary infective cause, provided that life is sufficiently prolonged for such a termination to take place, because in many instances death is produced earlier by compression on account of the unyielding character of the cranial vault, resulting in fatal compression of the brain which annihilates the essential physiological functions of the brain.

In the treatment we will briefly refer to the importance of establishing free drainage as a prophylactic measure; and the resort to all measures which are known to effect thorough local disinfection. It has been found, for instance, that it is not injurious to apply even caustics to the brain, so you are justified in cases of suspected infection after an injury to resort to local disinfection by thoroughly cleansing the wound and its vicinity, to be followed by the application of a ten per cent. solution of chloride of zinc to the wounded surfaces, and thorough and prolonged irrigation with a one-tenth per cent. sublimate solution—taking special precautions that these agents must be brought in contact with the entire surface which has been exposed to infection. The prophylactic treatment is of the greatest importance, while the curative is by general consent considered as *nil*. The life of the patient in all cases of compound fracture is, as it were, in your own hands. If the inflammation has taken place at the seat of traumatism before the patient has come under your care, the most conscientious application of all antiseptic precautions is

called for in all cases of wounds of the scalp and compound fractures of the skull, as furnishing the only guarantee against traumatic meningitis. The case is usually beyond hope of recovery after the infective inflammation has invaded the subjacent meninges. In a recent case before you touch the wound you must disinfect your hands with a strong solution of sublimate. To ascertain the extent of the injury, expose the upper portion of the wound by incising the scalp freely, and if there is any doubt as to the aseptic condition of the exposed meninx, you should disinfect it, being careful, at the same time, to arrest all hemorrhage, so that the formation of a subsequent blood-clot may not act as a favorable culture soil for any remaining germs. Prevent the accumulation of wound products by establishing free drainage of the subcranial space.

The old-time remedy, venesection, needs to be mentioned, not as a curative measure, but as a potent prophylactic remedy to modify reaction after an injury to the brain. When you are dealing with a case of contusion or concussion of the brain—injuries which are necessarily followed by a certain amount of reaction, if you can select the time when reaction is to be reëstablished to modify intravascular pressure, you may prevent undue engorgement by the use of timely and well-directed measures; consequently, at that stage, when cerebral irritation manifests itself by severe headache with a bounding pulse, if there are no contraindications, you are fully justified in resorting to the free use of the lancet as the most potent agent until you have brought the heart's action under your control. With a view of still further modifying the intracranial circulation, resort early to prolonged use of applications of ice to the head, which is particularly useful in this locality, because the vessels being located so near the surface, the ice has a direct effect in contracting the smaller vessels, thus

diminishing in direct manner the blood supply to the brain. This agent is not only effectual in modifying the circulation, but at the same time it has a prompt effect in reducing the temperature. The prolonged application of cold may also produce a salutary effect by arresting or retarding the reproduction of infective germs. Albert has well said that in all cases of injury to the skull and brain, you are expected to do something. He says there is no harm in the application of cold water, but when inflammation is threatened, it should always give way to the application of an ice-bag as one of the most potent means to meet the urgent indications. The beneficial effects of cold in modifying the cerebral circulation, in diminishing symptoms of central irritation are well known, and should never be omitted in the prophylactic and curative treatment of traumatic meningitis. In all cases of wound infective diseases we should aid the efforts of nature to eliminate infective germs by inducing an artificial gastro-intestinal catarrh, and for this purpose it is advisable to administer a large dose of calomel with the view of limiting also the cerebral congestion by inducing an artificial congestion in the alimentary canal.

I will next call your attention to the frequency of suppurative inflammation of the brain and its envelopes caused by preëxisting pathological processes independently of traumatism. In cases of suppurative osteomyelitis of the mastoid process, the vein which merges into the lateral sinus is often the medium of a direct extension of inflammation to the lateral sinus, the primary cause extending along that vessel by continuity and involving finally the sinus itself. It is that form of sinus-thrombosis and sinus-phlebitis that so frequently proves fatal in cases of inflammation of the internal ear and mastoid cells, producing, as a result, suppurative meningitis, abscess of the brain, and

pyæmia. Suppurative inflammation in the interior of the cranium has also not infrequently been observed as the result of caries of the cranial vault, taking place in a similar manner by continuity of the inflammatory process along the veins, extending, perhaps, to the longitudinal sinus, resulting in a sinus-thrombosis, sinus-phlebitis, and finally meningitis. The succession of pathological changes may, however, be the reverse, the process in the meninges extending by continuity of tissue directly to the sinus, producing sinus-phlebitis, which is in turn again followed by a sinus thrombosis. The symptoms indicating this lesion are those directly referable to a disturbance in the cerebral circulation. If a sinus has become suddenly obliterated either by extension of the thrombus to the sinus, or as a result of secondary sinus-thrombosis following sinus-phlebitis, a passive congestion on the venous side of the obstruction is a necessary and inevitable result; consequently there are symptoms indicative of cerebral congestion and fulness of the external veins which are contributory to the obliterated vessel. When the longitudinal sinus is the seat of obstruction, the veins of the forehead become distended; in case of sinus thrombosis of the lateral sinus, we observe dilatation of the vein over the mastoid process which perforates the bone and empties into the lateral sinus; obstruction in this particular vein being indicated *intra vitam* by local œdema in the region of the mastoid process. Please remember the fact that in case of sinus-thrombosis of the lateral sinus, following suppurative inflammation of the ear, one of the first evidences pointing to obstruction of the venous return from the lateral sinus is local œdema in the region of the mastoid process, and which is not attended by any evidences of superficial inflammation. Again, in case of sinus-thrombosis of the cavernous sinus an interference with the return of venous blood from the orbital region

takes place, indicated by an unusual prominence of the eyeballs, but as in this particular region the venous engorgement affects by its close proximity the nerves supplying the eye— the oculomotorius—you have, in addition, evidences of motor paralysis affecting the muscles of the eye. In thrombosis of the lateral sinus, when the thrombus increases in length in a central direction and reaches the jugular vein, the return of venous blood through this channel is obstructed. Gerhart says "this lesion is indicated by a preternatural emptiness of the external jugular vein on the affected side, due to a more easy return of the venous blood through the partially empty internal jugular." You will recognize the lesion by comparing the external jugular veins, locating the lesion on the side presenting a preternatural emptiness or collapsed condition of the vein. If, with this collapsed condition you have symptoms indicating a primary suppuration in the internal ear; and if at the same time you also have symptoms expressive of cerebral congestion, your diagnosis is almost positive: a thrombus in the lateral sinus.

In regard to the prognosis, just one word. If a sinus-thrombosis results from a direct injury to the sinus independently of traumatic infection, simply as the result of anatomical imperfection in the sinus itself arising from the traumatism, interfering with the circulation in the vessel, and the thrombosis is followed by a productive sinus-phlebitis, your prognosis is favorable, as a gradual obliteration of the sinus is not incompatible with a normal performance of the functions of the brain. If, on the other hand, a thrombus has occurred as the result of a suppurative sinus-phlebitis, or from infection from any other source, the thrombus does not become adherent; it does not serve as a medium in which the products of plastic proliferation find a favorable soil for growth and development, but it is an infective thrombus, and as a

result of that infection disintegration takes place, the thrombus breaks up, and produces embolism and pyæmia. Consequently in all cases of infective sinus-thrombosis and infective sinus-phlebitis, the result of infection either from osteomyelitis or wound infection, your prognosis must be guarded, as a fatal termination is inevitable.

In regard to treatment there is but little to say, inasmuch as most of these sinuses are not within reach of operative procedure. It has been suggested in the case of suppurative sinus-phlebitis of the superior longitudinal sinus to expose the sinus in order to remove the infected thrombus, with a view to prevent an extension of the infection to subjacent parts and to guard against pyæmia; but if you recollect the extent to which some of these channels may be blocked the measure appears hazardous to say the least. The inaccessibility of the cavernous sinus, and the importance of the structures in the immediate vicinity of the lateral sinus also preclude the advisability of operative interference. You will treat these cases, then, on the same principle as you would pyæmia. The most important element is the prophylactic treatment. In cases of suppurative inflammation of the mastoid cells do not postpone an operation until sinus-thrombosis has taken place, but resort to timely treatment by using the chisel to gain access to the infected structures, remove the infected tissues by *evidement* and ignipuncture, effect complete and thorough disinfection, and treat the wound as an infective one. These are the measures, when timely resorted to, which will prevent sinus-thrombosis in cases of suppurative inflammation of the cancellated tissue of the mastoid process. The same treatment should be adopted in all osteomyelitic processes wherever they involve the cranial bones.

Concussion.—We will next briefly consider the subject of concussion. By concussion of the brain I mean a sudden and complete annihilation of cerebral function as the result of an external application of force unattended by any recognizable anatomical tissue lesion. It is a perverted functional condition of the brain which results from a sudden application of force, producing a jar, as it were, of the brain substance, a condition which cannot be demonstrated in the brain, either microscopically or macroscopically; its essential feature consists in an altered relation between the molecular component parts of the brain, produced by a sudden commotion or jar. The complexus of symptoms which characterize this lesion appears immediately after an injury, and are expressed to us, in the first place, by a total loss of consciousness; the patient does not appear to recognize his condition; is totally ignorant of what has happened. Very soon after the accident he vomits, which appears as a mechanical act—it is not the kind of vomiting observed in cases of traumatic meningitis, but simply the evacuation of the contents of the stomach, which is undoubtedly an effort on the part of nature to restore the equilibrium of a disturbed circulation. The pulse is slow, from 50–60 per minute, soft and compressible. A peripheral paresis of the vasomotor system is indicated by extreme paleness of the cutaneous and mucous surfaces, clammy perspiration; respiration almost imperceptible; the respiratory movements being limited, imperfect, and slow. Acute cerebral anæmia following immediately upon concussion is indicated by an equal dilatation of the pupil; the paralysis, being partial and general, does not affect any particular muscular group, but all the voluntary muscles, the nerves of motion and sensation, as indicated by a depression of all of the cerebral functions; in other words, the prominent clinical features of concussion are:

unconsciousness; slow pulse; slow respiration; dilated pupils, with paresis of the sensory and motor nerves.

In making a differential diagnosis the only conditions which might lead to difficulty are the existence of contusion and compression. Concussion and contusion have been erroneously used as synonymous terms, but constitute two well-marked and distinct lesions, both from a pathological and prognostic standpoint. Concussion is usually an evanescent condition, not attended by any appreciable structural changes, while contusion signifies a laceration of the brain substance and some of the smaller bloodvessels, attended by extravasation of blood, followed by symptoms pointing toward a localized lesion in the brain. Difficulty in diagnosis arises from the fact that contusion is always attended by concussion; and the symptoms of concussion from the commencement may overshadow those due to the contusion. A differential diagnosis is only possible after the symptoms pointing toward concussion have disappeared. Compression due to traumatism, for instance after a depressed fracture of the skull, produces suddenly a diminution of space within the cranial cavity, and, if considerable, is almost sure to be attended by focal symptoms indicating compression of the brain. While the symptoms of concussion almost always subside within a few hours —or, at the most, within a day or two—the symptoms pointing to contusion or compression remain for a longer time, indicating a more serious injury to the brain.

In the treatment of concussion the most that is needed is physiological rest, absolute and complete, in the recumbent position, as this position favors a restoration of the disturbed circulation to its normal condition. If symptoms indicative of shock accompanying concussion present themselves, you may administer stimulants sufficient to counterbalance the depressing effect of the traumatism, favoring at the same time peripheral circu-

lation in the extremities by the application of external heat. The older English surgeons always resorted to repeated bleeding in cases of concussion, with a view to prevent subsequent inflammation. It is not necessary to state the result, perhaps, but a great many patients died, not from the concussion, but from the repeated venesections. Concussion uncomplicated by other lesions is only a temporary condition, and in the great majority of cases yields readily to nature's resources, consequently all that is necessary is to secure rest, and to rely on an expectant course of treatment; at the same time it is your duty to keep your patient under the closest observation until you have excluded the possibility of deeper and graver brain lesions.

One of the first symptoms frequently observed in cases of cerebral contusion are convulsions, local or general, according to the extent and location of the contusion. If the convulsive movements assume the form of a monospasm, the injury is usually located on the opposite side of the brain. Let me here call your attention to the fact that contusion does not necessarily occur always at the site of the external injury. If, for instance, the force has been applied to the right side of the head, and the paralysis or convulsive movements occur on the same side, it is evident that the transmission of force through the brain substance has resulted in contusion of the brain on the opposite (left) side. This is an extremely important point to remember, when we consider the causative relation between a contusion of the brain and brain abscess, where it is of the greatest importance to localize with accuracy the seat of the lesion. It is a familiar fact that surgeons have often been disappointed in seeking for pus on the injured side in operations for abscess of the brain, when perhaps by transmission of force the contusion and subsequently the abscess have occurred on the opposite side.

In conclusion, let me again impress upon your minds the importance of treating even the most insignificant wounds of the scalp under the strictest antiseptic precautions, with a view of securing for your patients absolute protection against the disastrous consequences arising from traumatic infection. In cases of contusion of the brain make an early diagnosis and adopt timely measures which will prevent undue vascular engorgement, and, to a certain extent, extravasation at the point of contusion. In concussion of the brain be careful to eliminate the existence of contusion by watching the course of the lesion diligently until a sufficient time has elapsed, when a positive differential diagnosis between concussion and contusion can be made; and after you have fully satisfied yourself that you are dealing with a case of concussion complicated by contusion, adopt measures which will prevent microbic invasion of the contused area, and which will, at the same time, moderate the vascular engorgement at the seat of contusion.